Case reports

Intra-A-type variation of WPW syndrome

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A patient with type A WPW syndrome and variation in the QRS morphology is described.

The WPW syndrome has been traditionally divided into two major types, though on the basis of the modern electrophysiological research this classification is becoming of less value. These types have been designated as type A and type B, depending on the orientation of the vector of the initial delta-wave in lead V1 (Rosenbaum et al., 1945). Variation between the A and B type conductions has been described in the same individuals (Matter and Hayes, 1964; Ramachandran, 1972; Lunel, 1972). However, remarkable variation of conduction seems also to be possible within a single major WPW type and during unchanged sinus rhythm, in addition to the well-known 'concertina effect' seen in tachyarrhythmias.

An intra-A-type variation of WPW syndrome occurred in an otherwise healthy man described in this report. The variant type was transiently present only after treatment of a paroxysm of fast atrial fibrillation with electrical cardioversion. This kind of striking variation of the QRS morphology in the standard leads, which initially focused our interest, does not appear to have been reported earlier.

Case report

Clinical course

The patient was a 22-year-old man who had suffered from occasional short attacks of rapid pulse in his childhood, but was otherwise healthy. He was admitted to another hospital because of chest pain which followed a sudden onset of rapid irregular heart beat. These symptoms had appeared when climbing upstairs. On admission his general condition was good. No signs of cardiac failure were present, the arterial blood pressure measured 140/80 mmHg (18.6/10.6 kPa), and the pulse rate was irregular, 140 to 150 beats/min, but showed a conspicuous pulse deficit. A presumptive diagnosis of ventricular tachycardia was made, however, and ligno-caine was administered as a loading dose of 100 mg i.v., followed by a continuous i.v. drip (2 mg/min). Since no effect was observed during several hours, successful electrical conversion was made with 100 J synchronized direct current. Propranolol 20 mg four times daily was later given as a prophylactic antiarrhythmic treatment, when the WPW syndrome was established as a cause of the tachyarrhythmias. With this treatment the patient has been completely free of bouts of tachycardia.

Electrocardiograms

A) **Admission** The 12-lead electrocardiogram recorded on admission displays an irregular tachycardia with the ventricular rate varying between 200 and 250 beats per minute (Fig. 1). The QRS complexes are predominantly widened to 0.12 s and mostly of the same configuration. The activation front is directed to the right (ÂQRSf +200°) in both the frontal and horizontal planes. The major forces are thus directed anteriorly, superiorly, and rightward. The electrocardiogram may thus be explained as a fast atrial fibrillation and abnormal ventricular conduction with its starting point in the posterolateral region of the ventricles. However, in leads III, V2, and V5 there are a few QRS complexes in a series of 2 to 5 which have quite narrow deflections of 0.08 s, appearing at a rate of 185 a minute. In fact, such QRS complexes of normal width never occurred later during sinus rhythm. While of normal width and of normal general shape, these transient beats showed minor terminal delays of activation.

B) **After electrical cardioversion** (Fig. 2, p. 768) The rhythm restored with a single 100 J shock shows sinus tachycardia. The PQ interval is 0.13 s and QRS duration 0.12–0.13 s. The P wave does not show any variation in its shape. The major ventricular activation forces are directed in the frontal plane upwards (−80°), turning then to the left, and in the horizontal plane anteriorly (+100°). The delta wave of the WPW pattern is clearly visible in both planes, being positive
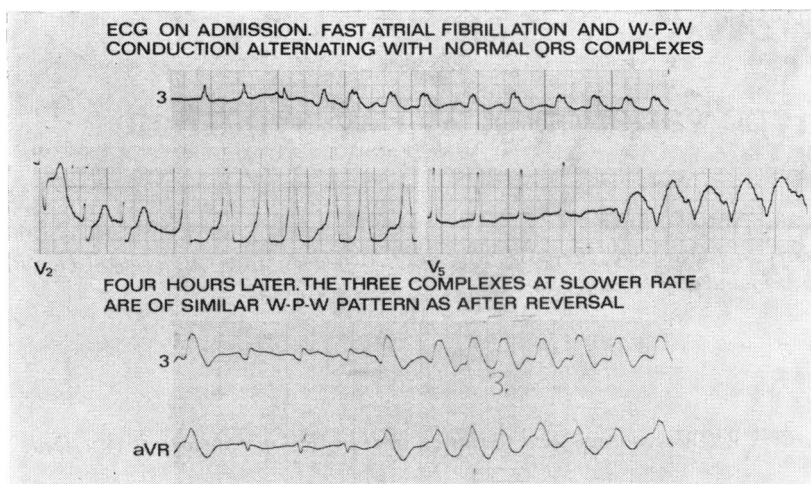
FIG. I   *Electrocardiogram recorded on admission.*

from V1 to V5 but inconspicuous in V6. In the frontal plane the delta wave orientates upwards and slightly leftwards. Thus the initial activation focus would be situated in the posterior regions of the heart.
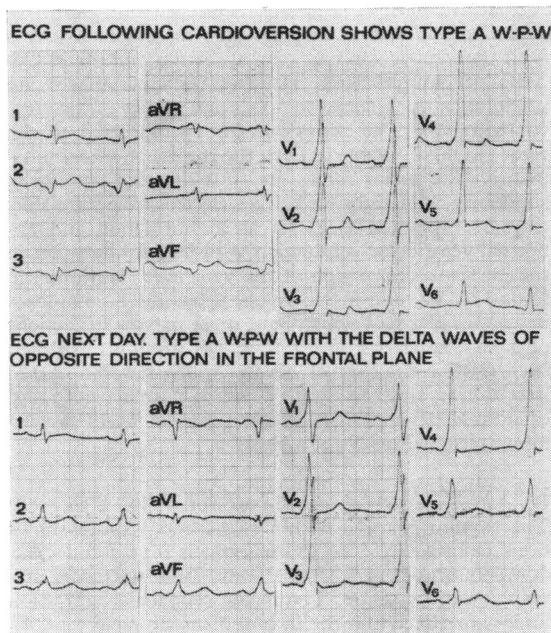
C) **Basic electrocardiogram** (Fig. 2, bottom).   The electrocardiogram recorded next day is similar to all later control records during the next 6 months. The P

FIG. 2   *The serial electrocardiograms reveal an intra-A-type variation of Wolff-Parkinson-White syndrome.*

wave is again similar to that in the immediate post-conversion rhythm. The PQ interval is now slightly shorter and QRS broader, i.e. 0.11 and 0.13–0.14 s. The main QRS vector is directed in the frontal plane roughly opposite to that of the postconversion time. The ÂQRSf is between +60° and +70° both for the delta wave and also for the main forces. In the horizontal plane, the main forces are similar to those seen before in the praecordial leads, showing minor variations only. Though the delta wave is positive again from V1 to V5 as before, there are two differences. Now the delta wave in V6 is also positive and also the delta waves are clearly broader. Thus, the initial activation focus would be located again in posterior basal regions, though with a slightly different insertion of the accessory pathway.

Discussion

In spite of the striking differences of the QRS complexes noted in the limb leads, both the electrocardiogram recorded after cardioversion (B) and that representing the basic rhythm (C) comply with the criteria of the major type A WPW syndrome. Changes in the main QRS vector in WPW syndrome (in the present patient in opposite directions in the frontal plane) are known to occur during the transition from sinus rhythm to atrioventricular rhythm. Moreover, it has been stressed that there is a high frequency of supraventricular ectopic foci in patients with pre-excitation syndrome (Sherf and James, 1969; Hindman, Last, and Rosen, 1973). This seems, however, not to be a likely explanation of the present case since the shape and size of the P waves are exactly the same in both electrocardiograms.

The most probable explanation of the electrocardiographic findings described above would be

the presence of two bypassing Kent type bundles near each other in posterolateral regions of the left ventricle. This conclusion is supported by both the vector analysis of the initial force as well as by the activation patterns documented by Boineau *et al.* (1973) in their thorough analysis of WPW conduction. It is also possible that one Kent bundle is accompanied by one septal Mahaim bundle (B), besides the normal atrioventricular node. Recent histological examinations of the heart in the WPW syndrome have revealed a sound anatomical basis for the occurrence of several accessory atrioventricular myocardial fibres, transversing gaps in a discontinuous annulus fibrosus (Lunel, 1972). This finding is nicely supported by direct mapping of the activation wave fronts on the epicardium (Boineau *et al.*, 1973). It is suggested that these Kent type fibres transmit impulses varyingly and thus are a cause of the A and B type variation in ventricular activation. The James–Mahaim hypothesis has so far remained less firmly documented (Moore, Spear, and Boineau, 1973).

Boineau and his co-workers (1973) have recently re-examined the electrocardiographic typing indifferent insertions of the anomalous connexion. They related the standard 12-lead electrocardiograms to the recorded mapping of activation sequences on the surface of the heart. According to their data electrocardiogram C of our patient would best resemble the type 4 in their model, i.e. lateral left ventricular pre-excitation, and electrocardiogram B again faithfully resembles type 3, a more posterior insertion. Notably, both these two possible Kent bundle locations 3 and 4 are in the posterolateral region, as deduced too by means of the vector analysis in our patient. They too show similar V-lead type A complexes but have opposite directions of activation in the frontal plane.

That the cause of the varying frontal plane QRS axis is related to different posterolateral accessory pathways close to each other has not been established. The posterior insertion of the bypass tract (electrocardiogram B, type 3), results in epicardial breakthrough at the posterior atrioventricular margin on the left side of the septum. The activation of these inferior areas is also first to be completed, before that of the anterior and superior regions. The pre-excitation impulse may be thought also to invade the posterior fascicle of the left bundle-branch, resulting in its early retrograde activation. In any case large posteroinferior muscle masses are the earliest areas activated, with a consequent left axis deviation caused by large superiorly oriented forces. In a more lateral insertion (electrocardiogram C, type 4) the frontal plane axis remains normal; further the anomalously activated muscle areas tend to

remain smaller, perhaps without retrograde bundle-branch activation. The slight differences in the PQ and QRS durations in our patient are in fact opposite from those that might be expected, because lateral activation is usually more delayed and produces less pre-excitation than the medial sites (Boineau *et al.*, 1973). These assumptions based on the time intervals alone, however, may be misleading if conduction velocities of different tracts have been altered for instance by the drug treatment.

The few normal-shaped QRS complexes observed during the tachyarrhythmia may represent a temporary complete failure of both accessory tracts caused by excessive firing because of atrial fibrillation. In fact, sudden Mobitz 2 type block best characterizes the electrophysiological properties of the anomalous connexion (Roelandt *et al.*, 1973). This allows normal conduction through the atrioventricular node. Similarly, the QRS morphology dominating during the tachyarrhythmia may result from varying ventricular conduction.

Cardioversion and/or antiarrhythmic drugs may have blocked one of the accessory tracts, i.e. that which dominates in mediating the basic rhythm. Different refractory times of the tracts may have caused the temporary finding seen in electrocardiogram B. For instance, lignocaine depresses both muscle conduction and atrioventricular node in varying degrees. The above possibilities in generating variable conduction are further supported by finding that the different Kent bundles differ remarkably in their conductivity in the atrial pacing tests (Moore *et al.*, 1973).

To our knowledge, this kind of variation of ventricular activation within one major type of WPW syndrome has not been reported earlier. It need not be rare, however, as suggested by the recent histological evidence of adjacent Kent type fibres located in gaps in the annulus fibrosus. Continuous monitoring of patients during tachyarrhythmias and immediately thereafter may show that shifts from one 'subtype' to another are more common than is believed.

References

Boineau, J. P., Moore, E. N., Spear, J. F., and Sealy, W. C. (1973). Basis of clinical-ECG variations in right and left ventricular pre-excitation: a unitary concept of WPW. In *Cardiac Arrhythmias*, p. 421. Ed. by L. S. Dreifus and W. Likoff. Grune and Stratton, Philadelphia.

Hindman, M. C., Last, J. H., and Rosen, K. M. (1973). Wolff-Parkinson-White syndrome observed by portable monitoring. *Annals of Internal Medicine*, 79, 654.

Lunel, A. A. V. (1972). Significance of annulus fibrosus of heart in relation to AV conduction and ventricular activation in cases of Wolff-Parkinson-White syndrome. *British Heart Journal*, 34, 1263.

Matter, B. J., and Hayes, W. L. (1964). Wolff-Parkinson-White syndrome. Report of a case with both type A and type B pre-excitation. *American Journal of Cardiology*, **13**, 284.

Moore, E. N., Spear, J. F., and Boineau, J. P. (1973). Recent electrophysiologic studies on the Wolff-Parkinson-White syndrome. *New England Journal of Medicine*, **289**, 956.

Ramachandran, S. (1972). Wolff-Parkinson-White syndrome. Conversion of type A to type B electrocardiographic changes. *Circulation*, **45**, 529.

Roelandt, J., Schamroth, L., Draulans, J., and Hugenholtz, P. G. (1973). Functional characteristics of the Wolff-Parkinson-White bypass. A study of six patients with His bundle electrograms. *American Heart Journal*, **85**, 260.

Rosenbaum, F. F., Hecht, H. H., Wilson, F. N., and Johnston, F. D. (1945). The potential variations of the thorax and the esophagus in anomalous atrioventricular excitation (Wolff-Parkinson-White syndrome). *American Heart Journal*, **29**, 281.

Sherf, L., and James, T. N. (1969). A new electrocardiographic concept: synchronized sinoventricular conduction. *Diseases of the Chest*, **55**, 127.